0195-9131/87/1904-0368$2.00/0
MEDICINE AND SCIENCE IN SPORTS AND EXERCISE
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Vol. 19, No. 4
Printed in U.S.A.

Sprint training effects on muscle myoglobin, enzymes, fiber types, and blood lactate

IRA JACOBS, MONA ESBJÖRNSSON, CHRISTER SYLVÉN, INGEMAR HOLM, and EVA JANSSON

Department of Clinical Physiology, Karolinska Hospital, Stockholm S-104 01, SWEDEN and Defence and Civil Institute of Environmental Medicine, P.O. Box 2000, Downsview, Ontario, CANADA M3M 3B9

ABSTRACT

JACOBS, I., M. ESBJÖRNSSON, C. SYLVÉN, I. HOLM, and E. JANSSON. Sprint training effects on muscle myoglobin, enzymes, fiber types, and blood lactate. *Med. Sci. Sports Exerc.*, Vol. 19, No. 4, pp. 368–374, 1987. The purpose of this study was to determine if changes in intra-muscular myoglobin concentration accompany histochemical and enzymatic adaptations to supra-maximal exercise training. Subjects were assigned to either a training group ($N = 11$), who trained 2 to 3 times weekly for 6 wk, or a control group ($N = 6$). Training progressed from two 15-s and two 30-s "all-out" sprints on a cycle ergometer during week 1 to six 15-s and six 30-s bouts per session during week 6. The Wingate test was performed before and after the 6 wk, but performance variables were not changed in either group. In the training group, peak lactate after the Wingate test was significantly higher after training. No significant changes in enzyme activities, myoglobin concentration, or fiber-type frequency were observed in the control group. In contrast, in the training group, the percent fast twitch oxidative fibers increased, myoglobin decreased, and both citrate synthase and phosphofructokinase activities increased ($P < 0.05$). The results suggest that muscle myoglobin concentration is not increased by 6 wk of supra-maximal exercise training and that such training induces cellular adaptations without accompanying performance changes. Alternatively, the Wingate test is not a sensitive test of adaptations to the training.

ANAEROBIC EXERCISE, WINGATE TEST, ENERGY METABOLISM, PHOSPHOFRUCTOKINASE, CITRATE SYNTHASE

The extent of peripheral adaptations to physical training depends on the intensity of training. It has been well-documented that endurance training induces enzymatic adaptations which facilitate energy derivation via oxidative phosphorylation [(9, 10), for review see (36)]. Such adaptations may also be accompanied by various histochemical and morphological changes of the muscle fiber (10, 43). Far fewer studies have documented peripheral adaptations to supra-maximal exercise training (4, 15, 16, 37, 45). Such exercise is by definition an intensity that exceeds the capacity of the musculature to derive energy via oxidative phosphorylation. Therefore, the derivation of energy must occur mainly from both glycogenolysis leading to lactate production, and utilization of local adenosine triphosphate (ATP) and creatine phosphate stores.

Theoretically, oxidative phosphorylation can contribute to energy derivation for supra-maximal exercise through the intra-muscular stores of myoglobin. These stores provide oxygen in close proximity to the mitochondria from a source which is not immediately dependent on the rate of oxygen uptake and transport when exercise is initiated. This is exemplified by the myoglobin levels in the skeletal musculature of diving mammals, which are several times greater than those of land mammals (46). In the anaerobic environment of an underwater dive, lactic acid accumulates only after myoglobin is deoxygenated, requiring 5 to 10 min in seals (38). These observations and other reports of higher muscle myoglobin levels in man and animals chronically exposed to high altitude, relative to their sea level counterparts [for review see ref. (46)], suggest that an adaptation to chronic increases in oxygen demand may be an increase in myoglobin concentration.

Consistent with this hypothesis, endurance training of animals results in an increased intra-muscular myoglobin concentration (14, 32). In contrast, human endurance training (24, 39) and detraining (29) studies demonstrated that myoglobin concentration does not change, although other peripheral adaptations were observed, such as marked changes in muscle enzyme activities. It was therefore hypothesized that the large oxygen deficits associated with supra-maximal exercise training, in contrast to endurance training, might stimulate the myoglobin-oxygen binding mechanism in a manner analogous to that reported for diving mammals, resulting in an increased myoglobin synthesis.

The specific purpose of the present study was to determine if changes in intra-muscular myoglobin concentration would accompany the changes in muscle

Submitted for publication May, 1986.
Accepted for publication February, 1987

88-00355/

histochemical and enzymatic characteristics which have previously been documented to occur as adaptive responses to supra-maximal exercise training.

METHODS

Subjects. Seventeen students at a college for sports and recreation instructors volunteered to participate after being fully informed of the details, risks, and possible discomforts associated with the experimental protocol. The study was approved by the Ethics Committee of the Karolinska Hospital, and written informed consent was obtained from all subjects. In accordance with the subjects' own choice, they were assigned to either the training group [(Tr) 7 males and 4 females] or the control group [(Con) 4 males and 2 females]. An analysis of variance showed that age, height, and weight for both groups were similar and averaged (range) 22.3 yr (22 to 29), 163 cm (158 to 189), and 70.0 kg (54 to 92), respectively.

Performance test. The Tr subjects performed the Wingate test (WT) on the day prior to the initiation of training and 2 d after the 6-wk training period. The Con subjects performed the WT twice, with a similar intervening period of 6-wk. The WT was performed on a mechanically braked cycle ergometer (Cardionics®, Skärholmen, Sweden) as described previously (19, 20). A sensor-microprocessor assembly counted flywheel revolutions with a resolution of 0.3 revolutions and, based on the individual resistance (75 $g \cdot kg^{-1}$ body weight), the flywheel progression per pedal revolution (6 m), and the elapsed time, the average power output was calculated and printed automatically each 5 s for the duration of this 30-s test. The following performance variables were calculated: peak power (i.e., the highest 5-s power output); mean power (i.e., the average power during the 30-s duration); and a fatigue index calculated as the difference between peak power and the last power output, expressed relative to peak power (i.e., the greater the fatigue index, the greater was the drop in power).

Blood lactate. On both test occasions 25 μl blood were sampled from a finger tip 1 to 4 min, respectively, after the WT, while the subject remained passively seated on the cycle. The blood was immediately deproteinized in perchloric acid, refrigerated, and assayed for lactate concentration (25) within 1 wk. The coefficient of variation for the lactate assay was 5% (25).

Training. Training was performed on mechanically braked cycle ergometers (Cardionics or Monark) an average of 2.5 $times \cdot wk^{-1}$ for a total duration of 6 wk. For purposes of comparison, the training model was similar to that reported by Grodjinovsky et al. (12). During the first week, a training session consisted of two 15-s and two 30-s "all-out" cycle sprints against a resistance of 75 $g \cdot kg^{-1}$ body weight. Each week thereafter, additional 15-s and 30-s bouts were added to each session so that by the last week, six 15-s and six 30-s sprints were performed per training session. There was an uncontrolled rest interval of 45 s between consecutive 15-s sprints and a rest interval of 15 min between the 30-s sprints. Training logs were maintained by each subject documenting that they did adhere to the required training frequency.

Muscle biopsies. The needle biopsy technique (2) was used to sample muscle tissue from the m. vastus lateralis on the day before training commenced and 2 d after the final training session. The Con subjects were also biopsied twice with a similar intervening period of 6 wk. The same investigator performed all biopsies and took two tissue samples from a single incision. One sample was immediately frozen in liquid nitrogen and then stored at −80°C until assayed. This sample was freeze-dried and dissected free of connective tissue and dried blood under a light microscope. The activities of citrate synthase (CS) (6), phosphofructokinase (PFK) (6), and total creatine kinase [Boehringer-Mannheim Kit, (Mannheim, West Germany) *N*-acetyl cysteine-activated creatine kinase] were assayed with enzymatic fluorometric or spectrophotometric techniques. Intramuscular myoglobin concentration was determined by a radioimmunological assay (28). Each tissue sample was divided into three pieces, and each of these was assayed in duplicate. Pre- and post-training samples from each subject were assayed in the same batch. The coefficients of variation, expressed as the SD of the difference between double determinations, relative to the total mean value, are: 7% for myoglobin (23); 20% for creatine kinase (41); 18% for PFK (6); and 12% for CS (6).

The second tissue sample was mounted in an embedding medium and frozen in isopentane cooled in liquid nitrogen. Serial cross-sections were stained for myofibrillar ATPase (31) after pre-incubation at pH 10.3, 4.6, and 4.3 (3), and the fibers were classified as slow twitch (ST) or fast twitch (FT) a, b, or c. Only serial sections with more than 200 fibers were used for the determination of the relative fiber-type distribution. A serial section was also stained for NADH-tetrazolium reductase (30) and was used for the calculation of mean fiber area, with a cutting and weighing method described by Thorstensson (44). The relative area occupied by each fiber type was calculated according to the formula described by Tesch (42). The pre- and post-training biopsy samples for each subject were stained together.

Statistics. Data are expressed as mean ± SD unless stated otherwise. The significance of inter-group differences prior to training was determined with an analysis of variance. Intra-group differences between the pre- and post-training mean values were calculated with a two-tailed Student's *t*-test. Statistical significance was accepted at the 0.05 level.

RESULTS

Pre-training group comparisons. With the exception of peak power·kg^{-1} body weight which was greater in the Tr group, the Tr and Con groups did not differ significantly in their performance of the WT prior to the training period. Group mean values were similar with regard to blood lactate values after the WT, muscle fiber characteristics, muscle enzyme activities, and myoglobin concentration.

Performance. The 6 wk of training did not cause significant improvements in performance of the WT (Table 1). There was, however, a subjective feeling by the subjects of an enhanced ability to perform repeated bouts of the WT. Most subjects complained of nausea and exhaustion following the first performance of the WT. All Tr subjects reported that these feelings had dissipated by the last training week when six bouts of the WT could be performed per training session.

Blood lactate. Peak lactate concentration after the WT was increased after 6 wk in 9 of the 11 Tr subjects; the mean values for the Tr group were 11.4 ± 1.0 and 12.5 ± 1.3 mmol·l^{-1} before and after training, respectively. Significantly higher lactate levels after training were evident at each of the 4 min after exercise (Fig. 1).

Fiber types. The fiber-type distribution in the Con group was similar before and after the 6 wk. In contrast, the %FTa fibers increased in the Tr group from 31.9 ± 8.0% before training to 39.1 ± 8.0% after training (P = 0.008) (Fig. 2). This was accompanied by a decrease of the ST fiber percentage from 57.7 ± 16.6% to 48.3 ± 9.3% (P = 0.087). No significant changes were observed in either group for FT or ST fiber areas, percent area occupied by the ST or FT fibers, nor for the ratio FT/ST fiber area (Table 2).

Enzymes and myoglobin. The results of the enzyme and myoglobin assays are shown in Table 3. While no significant changes were observed in the Con group, 7 of 11 Tr subjects increased CS activity (P = 0.030) and 8 of 11 increased PFK activity (P = 0.011) after train-

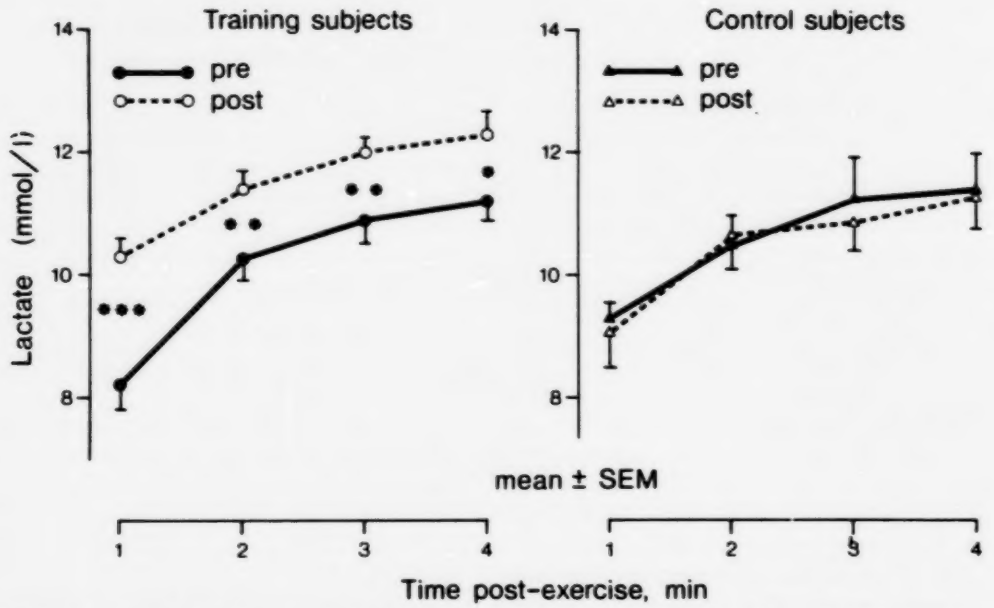

Figure 1—Blood lactate concentrations after performance of the WT. The significance of the pre- vs. the post-training comparisons are shown: * = $P < 0.05$; ** = $P < 0.01$; *** = $P < 0.001$.

TABLE 1. Performance variables measured during the WT for the Tr and Con groups.

		Tr		Con	
Variable	Units	Pre-	Post-	Pre-	Post-
Peak power	W	801 ± 163	789 ± 189	775 ± 164	828 ± 162
	W·kg^{-1}	12.0 ± 1.3	11.9 ± 1.5	10.8 ± 0.8	11.6 ± 0.9
Mean power	W	605 ± 146	613 ± 153	586 ± 115	600 ± 127
	W·kg^{-1}	9.3 ± 1.6	9.2 ± 1.2	8.2 ± 1.2	8.4 ± 1.0
Power decrease	%	42 ± 7	40 ± 7	45 ± 10	48 ± 5

Data are mean ± SD.

ing. Muscle myoglobin concentration was significantly decreased in the Tr group after training ($P = 0.012$). Nine of the 11 subjects had a decreased myoglobin level, one subject remained at the same level, and only one subject demonstrated an increased level after training.

DISCUSSION

The main finding in the present study was that intramuscular myoglobin concentration was not increased, but was decreased, after 6 wk of high intensity exercise training. No other studies have reported the effects of sprint training on human intra-muscular myoglobin levels, so the possibility exists that a longer duration of training may have induced increases in myoglobin. The apparent lack of myoglobin adaptation in the present study is similar to the endurance training study of Svedenhag et al. (39), who reported no changes in myoglobin concentration after 8 wk of training, in spite of marked increases in the activity of mitochondrial enzymes and $\dot{V}O_{2max}$. In another longitudinal study, Nemeth and Lowry (29) reported that 12 wk of detraining in a highly trained athlete did not affect the myoglobin level. Similarly, Jansson et al. (24) found no difference in the intra-muscular myoglobin concentration between competitive cyclists and sedentary subjects.

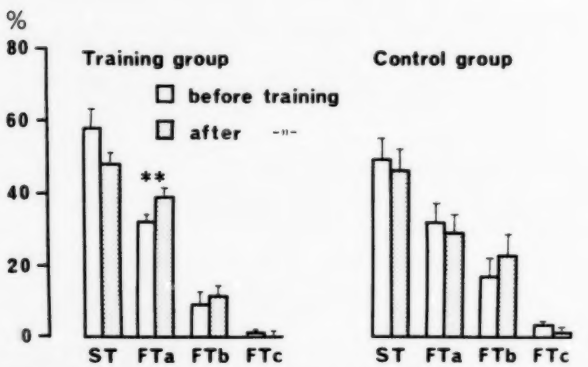

Figure 2—The frequency of various muscle fiber types in m. vastus lateralis tissue samples obtained before and after training. ** = $P < 0.01$ for the significance of the difference between the pre- and post-training value. Values are mean ± SE.

In contrast to the relatively low intensity endurance training examined in these latter studies, the supramaximal exercise used as the training stimulus in the present study results in a large oxygen deficit because the generated power outputs exceed maximal aerobic power (18). Reports of high rates of muscle lactate accumulation and depletion of endogenous creatine phosphate stores (19, 20) demonstrate that significant amounts of energy are derived from metabolic pathways other than oxidative phosphorylation during the WT. We hypothesized that repeated formation of such large oxygen deficits would provide an adequate stimulus for skeletal muscle myoglobin synthesis. Millikan (27) demonstrated that the deoxygenation of myoglobin is well underway in less than 0.2 s after the onset of muscular contraction. It therefore seemed reasonable to speculate that myoglobin would respond to training as do other intra-muscular proteins, such as the marker enzymes of energy metabolism pathways.

The finding of a significant decrease in muscle myoglobin after training was unexpected and cannot be adequately explained based on the present data. One possible explanation may be associated with muscle cell damage which occurs after acute hard exercise. The membrane ruptures, which can be observed with electron microscopy (8), indicate that leakage of myoglobin out of muscle cells is a distinct possibility. Further support for this possibility is found in the work of Sylvén and Benz (40), who demonstrated that plasma myoglobin levels increase after muscle cell damage, and by a report of greater than five-fold increases in serum myoglobin concentration after an acute bout of high

TABLE 3. Skeletal muscle enzyme activities and myoglobin concentration for the Tr and Con groups, expressed relative to dry muscle weight.

	Tr		Con	
	Pre-	Post-	Pre-	Post-
CS ($\mu kat \cdot g^{-1}$)	0.924 ± 0.133	1.036 ± 0.066*	0.942 ± 0.103	0.937 ± 0.120
PFK ($\mu kat \cdot g^{-1}$)	1.10 ± 0.17	1.28 ± 0.20*	1.20 ± 0.15	1.11 ± 0.15
CK ($\mu kat \cdot g^{-1}$)	383 ± 75.0	351 ± 57.4	459 ± 59.8	434 ± 45.6
Myoglobin ($mg \cdot g^{-1}$)	17.6 ± 4.3	15.3 ± 3.0*	18.6 ± 3.7	18.3 ± 3.4

Data are mean ± SD.
* $P < 0.05$.

TABLE 2. Muscle fiber area characteristics.

		Tr		Con	
Variable	Units	Pre-	Post-	Pre-	Post-
ST fiber area	μm^2	4,705 ± 965	5,157 ± 1,075	4,882 ± 556	4,804 ± 982
	% of muscle	53 ± 17	44 ± 10	41 ± 12	42 ± 17
FT fiber area	μm^2	5,561 ± 1,635	5,900 ± 1,701	5,403 ± 1,188	5,467 ± 980
	% of muscle	47 ± 17	56 ± 10	59 ± 12	58 ± 20
FT/ST area		1.17 ± 0.20	1.14 ± 2.09	1.10 ± 0.17	1.17 ± 0.32

Data are mean ± SD.

intensity exercise (35). Such a leakage could counteract any intra-muscular elevation in myoglobin concentration caused by the training. A calculation, however, of the total amount of myoglobin that would have to leave the leg musculature during the training period to account for the decrease observed in the present study yields 20×10^9 ng. A normal plasma myoglobin concentration is approximately 50 $\mu g \cdot l^{-1}$ (35); thus, it is unlikely that 20×10^9 ng could be released to the plasma without myoglobinuria, of which there were no macroscopic signs in the present study.

An alternate explanation for the lack of increase in myoglobin in humans after physical training may be associated with the rather high pre-training myoglobin concentrations in human skeletal muscle (20 $mg \cdot g^{-1}$), relative to those of rat muscle (8 to 14 $mg \cdot g^{-1}$). Perhaps myoglobin exists in sufficient quantities in untrained human skeletal muscle to meet the physiological demands of both endurance and sprint exercise.

The second finding of note in the present study was the significant increase in the frequency of FTa fibers in the Tr group after training. The 8% increase in FTa fibers was accompanied by a commensurate decrease in ST fiber percentage, although the latter change failed to achieve statistical significance. These changes may also partially explain the decreased myoglobin levels because ST fibers contain more myoglobin than FT fibers (23, 29); the observed decrease may have been caused because the biopsy sample after training contained more ST fibers than that taken before training.

The biopsies in the present study were obtained from both groups approximately 1 h after performance of the WT, before and after the training period. It has been suggested that supra-maximal exercise may induce temporary changes in the myofibrillar ATPase reaction which result in an apparent decrease in the percentage of ST fibers if a biopsy is taken soon after exercise (1, 11). These and other methodological errors could account for the observed change in the FTa percentage, but both the Tr and Con groups were biopsied by the same investigator at similar intervals after performance of the WT, yet significant fiber type changes were only observed in the Tr group.

The FTa subgroup of FT fibers is characterized by relatively high activities of both glycolytic and oxidative enzyme activities compared to the other FT subgroups [for recent review, see ref. (36)]. Therefore, the observed increase in FTa fibers is consistent with the fact that Tr exhibited increases in both CS and PFK activities. Although cross-sectional comparisons of subjects with widely different physical activity backgrounds suggest that individuals with good sprint abilities have a relatively high percentage of FT fibers, the present results should be interpreted with care in light of the recent review by Saltin and Gollnick (36), who concluded that physical training probably does not cause conversions of ST into FT fibers or the reverse. Other studies which have utilized supra-maximal exercise training have not demonstrated ST to FT fiber conversions (4, 16, 37), although Jansson et al. (22) did report significantly less ST fibers in the vastus lateralis of four distance runners after they suddenly switched to "anaerobic" training for about 10 wk.

A third finding in the present study was the lack of performance changes after training, in spite of significant metabolic adaptations to the training. The increase in peak blood lactate levels after exhaustive exercise is a well-established adaptation to high intensity exercise training (16, 26, 37). Although acute WT performance causes an approximately 10% decrease in plasma volume (33), there is no support for the suggestion that supra-maximal exercise training causes a more marked decrease in plasma volume. Therefore, the higher peak lactate levels in the Tr group are not likely to be an artifact of different plasma volume shifts compared to the Con group. Taken together with the Tr group's 17% increase in PFK activity, there is evidence of an enhanced potential to derive energy via glycogenolysis or glycolysis after the training used in the present study. A mitochondrial enzymatic adaptation was demonstrated in the increased CS activity in the Tr group. The increased activities of both glycolytic and mitochondrial skeletal muscle marker enzymes confirm several previous reports where interval training with repeated sprints of a duration similar to those used in the present study resulted in similar enzymatic adaptations in the trained musculature (4, 16, 37).

Although the good test-retest reliability of the WT is well-documented (5, 7), longitudinal studies which document the sensitivity of the WT to training are scarce. In children, mean power generation during the WT was increased in subjects who trained using the WT as the training medium, as well as in subjects who engaged in interval training with repeated running sprints (12). Even endurance training, however, will elicit WT performance improvements in children (34); thus, the results may not be applicable to adults. In two studies using military personnel as subjects, mean power was increased, and muscular endurance was enhanced after circuit training programs (17, 21). The lack of measurable performance change in the present study could be attributed to the rather short duration of training and to the fact that training, although intense, was performed only twice weekly. Skeletal muscle cellular adaptations to endurance training precede measurable changes in maximal aerobic power (13), so it is not unreasonable to expect a similar time course to apply to "anaerobic," sprint training. Both the present results and those of Houston et al. (16) demonstrate that marked increases in peak blood lactate levels and activities of glycolytic enzyme markers can occur without significant changes in sprint exercise performance. An

alternative explanation is that the WT is insensitive to the training used in the present study.

In conclusion, the present study provides evidence that high intensity exercise training which elicits marked metabolic adaptations does not increase intramuscular myoglobin concentration. In conjunction with other studies which demonstrated similar results after endurance training, it should be emphasized that rat and human skeletal muscle differ in this regard. In addition, as is the case with adaptations to endurance training, enzymatic adaptations to supra-maximal exercise training probably precede changes in exercise performance. Further longitudinal training studies should be performed before it is assumed that the WT is sensitive to the effects of sprint training.

This study was partially supported by a grant from the Swedish Sports Research Council.

I. Jacobs is a Fellow of the American College of Sports Medicine.

Address for correspondence: Ira Jacobs, Dr. Med. Sc., Defence and Civil Institute of Environmental Medicine, P.O. Box 2000, Downsview, Ontario, Canada M3M 3B9.

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